

Short reports

Function of the diaphragm before and after plication

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Plication of the diaphragm increases lung volume and decreases paradoxical motion in diaphragm paralysis.¹ There are two theoretical mechanisms by which lung volume can be increased after plication. First, the paralysed diaphragm can develop a passive tension because of passive length-tension characteristics of the muscle. Plication may reduce diaphragmatic compliance so that passive tension develops more readily. The diaphragm would then act as a more effective pressure partition between the abdomen and thorax. This should result in increased transdiaphragmatic pressure during inspiratory manoeuvres after plication. Second, the increased lung volume after diaphragm plication may simply result in altered geometry of the diaphragm. A decrease in the area of apposition of the diaphragm may increase lung volume while still preserving the dome shape of the diaphragm.² Transdiaphragmatic pressure may not change after plication. In order to determine which mechanism is primarily responsible for the increase in lung volume after plication, we measured transdiaphragmatic pressure, pleural pressure, and abdominal pressure in a patient with severe bilateral diaphragmatic weakness before and after bilateral diaphragm plication.

Case report

A 17-month-old baby girl with an idiopathic peripheral neuropathy was admitted with hypercapnic respiratory failure. She had rapid shallow respirations. Although the chest increased in antero-posterior diameter during inspiration, there was a paradoxical inward motion of the abdomen. She required a tracheostomy and mechanical ventilatory assistance. The chest radiograph revealed elevated diaphragms and decreased lung volumes. Phrenic nerve conduction times were markedly prolonged. The diagnosis of severe bilateral diaphragm weakness was confirmed by transdiaphragmatic pressure measurements.

Severe diaphragm weakness and paradoxical movement of the diaphragm prevented weaning the patient from mechanical ventilation. Therefore, bilateral diaphragm plication was performed at 2 years of age. Both

diaphragms were elevated at surgery, the right more so than the left. Both phrenic nerves were noted to be thin. The central tendon of the diaphragm was identified. A row of sutures was placed Lembert fashion to invert the central tendon. Additional rows of Lembert sutures were placed until the diaphragm was held down in a fixed and semi-taut position. This procedure was performed on both hemi-diaphragms.

After plication, AP and lateral chest radiographs revealed diaphragms in a more normal position suggesting improved lung volumes. Although she could not be completely weaned from mechanical ventilation, she now tolerates over 30 minutes at a time without ventilatory support.

Methods

Transdiaphragmatic pressure (Pdi) is the difference between intra-abdominal pressure (Pab) and intrathoracic pressure (Ppl).³ It represents the measure of diaphragmatic function. Pdi was measured according to the method of Agostoni.³ Ppl was measured using an 8 centimetre balloon-tipped intraoesophageal catheter filled with 0.1 ml of air, positioned radiographically in the midoesophagus. The position was confirmed by the presence of cardiac oscillations in the Ppl tracing. Intra-gastric pressure (Pgas) was measured using an 8 centimetre balloon-tipped catheter filled with 1 ml of air. Pdi was measured directly by comparing Ppl and Pgas across a differential pressure transducer. Measurements were obtained continuously in the sitting position during tidal breathing. Maximal respiratory pressures were measured during maximal inspiratory manoeuvres against an occluded airway at FRC. In this patient maximal inspiratory efforts occurred during crying, and the highest pressures of several breaths were used. We assume that effort did not change appreciably during crying before or after surgery. In the same way Ppl and Pab were measured separately referenced to atmosphere.

Results

Overall lung volume increased after diaphragm plication as could be seen by comparing chest radiographs obtained at 25 cm H₂O (2.45 kPa) positive airway pressure, before and after surgery. Transpulmonary

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pressure at FRC was 1 cm H₂O (0.1 kPa) before and after plication. There was no change in transdiaphragmatic pressure (Pdi), pleural pressure (Ppl), or abdominal pressure (Pab) during tidal breathing or maximal inspiratory manoeuvres after plication. This is shown in the table. There was no change in tidal volume measured during spontaneous breathing before and after plication. However, dynamic pulmonary compliance improved slightly.

Discussion

The chest radiographs before and after surgery were obtained at end-inspiration at the same positive airway pressure. There was no change in the configuration of the chest wall after surgery. However, the diaphragms previously elevated, are in a more normal position after surgery. Although lung volumes were not measured in this patient, the changes in the chest film suggest that lung volume increased after plication.

There was no increase in transdiaphragmatic pressure (Pdi), pleural pressure (Ppl), or abdominal pressure (Pab) associated with increased lung volume after bilateral diaphragm plication. If increased lung volume was secondary to a pressure partition effect of the diaphragm because of decreased diaphragmatic compliance, then the increased passive diaphragm tension should separate intrathoracic pressure from intra-abdominal pressure. During inspiration, a pressure gradient would develop across the diaphragm (Pdi) because of a negative intrathoracic pressure generated by intercostal accessory muscles and decreased transmission to the abdomen. However, this did not occur. Therefore, the increase in lung volume after plication did not result from the diaphragm acting as a more effective pressure partition.

Kim and co-workers⁴ showed that Pdi is primarily determined by diaphragm muscle tension, rather than by geometrical configuration. The absence of a change in Pdi after plication in this patient suggests that a change in diaphragm tension has not occurred. Thus, the increase in lung volume must be caused primarily by changes in the geometric configuration of the diaphragm.

Gibson and co-workers⁵ showed that diaphragm paralysis decreases pulmonary compliance, possibly as a consequence of microatelectasis. A change in the geometrical configuration of the diaphragm and thorax may reduce microatelectasis, and thus increase pulmonary compliance.

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Table Measurements before and after bilateral diaphragm plication

	Tidal breathing (End-inspiration)		Maximal inspiratory (Occluded airway at FRC)	
	Before plication	After plication	Before plication	After plication
Transdiaphragmatic pressure (cm H ₂ O)	3.3 ± 0.1	4.2 ± 0.2	5.9 ± 0.3	4.9 ± 0.4
Intrapleural pressure (cm H ₂ O)	-7.7 ± 0.1	-6.3 ± 0.2	-9.8 ± 0.4	-8.4 ± 0.3
Intra-abdominal pressure (cm H ₂ O)	-4.4 ± 0.3	-2.1 ± 0.3	-3.9 ± 0.4	-3.5 ± 0.2
Respiratory frequency (breath/min)	42	45		
Tidal volume (ml)	29 ± 2	30 ± 1		
Transpulmonary pressure difference (cm H ₂ O)	9.6 ± 0.3	8.4 ± 0.5		
Dynamic compliance (ml/cm H ₂ O)	3.0 ± 0.2	3.6 ± 0.2		

Mean values ± standard error of the mean are shown.
1 cm H₂O equals 0.098 kPa.